ACARUS SCABEII

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THE acarus of scabies is a constant parasite of the human race. Every physician in his practice sees many cases yearly, but I doubt if the average physician is able to make a positive diagnosis by actual demonstration of the parasite more often than in one patient out of five. This inability to demonstrate the acarus is usually due to a misconception of the pathology of the lesions and the lack of knowledge as to where in the burrow the parasite is to be found. As we read our textbooks it seems a simple matter to locate and pick out the acarus and examine it under the microscope. However, I know of dermatologists who are never able to satisfactorily demonstrate the organism.

When one thinks about what happens in the burrow it is not difficult to accurately trace the parasite and remove it for demonstration. The majority of physicians in their search immediately open the vesicle and search for the parasite within its contents and are baffled when no parasites are found. They also attempt to find the parasite in the papular lesions on the skin of the forearms and abdomen where the search is very difficult, as it is only in the burrows that the organism can be seen and successfully recovered. In lesions on the wrists and hands, where burrows are always present, there should never be any failure in the demonstration of the acarus. The drawing that accompanies the article shows rather schematically the conditions and types of the burrows one most commonly finds.

When the burrow is examined, if it has been there for some time, one first sees the entrance end of the burrow with the skin exfoliated. This is a remnant of the old burrow and has been passed through several days before. We naturally do not find the acarus here. By following this we next see the tract of the burrow, which is filled with tiny black grains; these are made up of altered blood and the feces of the insect. The parasite is also not found here. Next we come to a vesicle with an inflammatory reaction, and here we search with very rare success for the parasite that is ordinarily not found therein. If we then carefully scrutinize the skin surrounding the burrow, we find a faintly inflamed tract passing out from the vesicle, usually on the opposite side, and by following it to its end we see a tiny white dot that resembles a scale in the skin. At this point there is no inflammatory reaction. Now if we take the tip of a knife—and I find a paracentesis knife the best for the purpose—and prick out this white body and set it into a drop of water, we find that we have recovered the acarus of scabies. The physician who carries out these steps of examination and searches for the white dot at the uninflamed extremity of the burrow has learned to successfully discover the parasite.

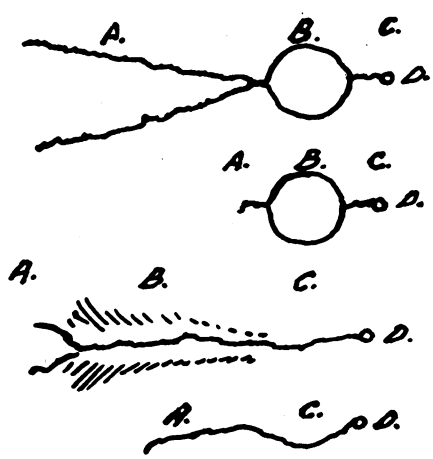

Fig. 1.—A. Old tract of entry. B. Vesicle or other type of reaction. C. Recent tract. D. Immediate position of acarus.

A brief consideration shows the cause of failure and the reason why the vesicle does not contain the parasite. We do not find the parasite at the beginning of the burrow for obvious reasons. Next, the vesicle is an inflamed reaction to the irritant action of the parasite and whatever bacteria may have been carried into the skin by its presence. The formation of a vesicle from irritation and the process of bacterial growth is of course slow, and probably twelve hours or more elapse before a vesicle is formed. Within this passage of time the acarus has continued onward and the vesiculation follows behind it at a definite interval. The acarus is found at the outermost extremity because it is there it is actively burrowing. The acarus also is found immediately below the surface layers of the skin, because it is not a deep worker, and the skin is sufficiently translucent to show its whitish body shining through. Lesions on the abdomen and arms of patients, unless they be infants, do not show definite burrows, as the acarus probably does not remain long in these areas of tender skin but burrows in, feeds, and comes out and then seeks a new location. In thickened skin the labor of entry is greater, so that it chooses to remain in the happy hunting ground of its burrow. When the parasite is found in the vesicle it is probable that more than one parasite has been present or has been caught in a resting stage or has lingered to lay eggs or carry out its amours.

In summarizing, then, do not look for the *Acarus scabeii* in the vesicle, but only where the white dot demonstrates its definite presence at the end of its burrow. In the absence of a burrow the diagnosis is necessarily made on the symptomatology and the distribution of the lesions.

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PSITTACOSIS

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THE State Board of Health has called attention to the existence of psittacosis in California. The following report may therefore be of interest:

REPORT OF CASE

CASE 5003.—Mrs. S., white, female, age fifty-one, weight about 187, height about five feet four and one-

half inches, rather overnourished, with fat over the abdomen rather excessive.

In June, 1929, the patient suffered from an attack of backache which she described as lumbago. A small renal calculus of right side was passed at that time, and the lumbago cleared up. Urine: Specific gravity, 1030; acid; albumen and sugar, negative; acetone and bile one plus; many amorphous crystals; pus and blood cells one plus; some epithelial cells.

On May 31, 1929, the patient complained of feet swelling, and loss of energy. Urine: Specific gravity, 1027; acid; albumen and sugar, negative; microscopically negative.

Patient came under observation for the attack here reported on January 2, 1932. Patient complained of abdominal pains and diarrhea (seven or eight stools a day), some cough, some distress in lower left chest. Temperature, 103.6 degrees; pulse, 110 to 120; respiration, 24 to 30. Patient had been sick for two days, and had had a chill followed by increase in temperature and by distress in lower left lung. Apparently the diarrhea developed after a cathartic. The skin was moist, and patient complained of sweating. There was marked disturbance of the mental faculties, a low delirium, and the patient found it rather hard to collect her thoughts.

A working diagnosis of respiratory infection by one of the hemolytic group was made. Some moist râles and decreased respiratory and voice sounds, and a slight area of dullness in the lower left lung were noted. At the end of the second day the left lung cleared, but the same picture appeared in the middle lobe of the right lung, just external to the nipple. Temperature and pulse remained about the same, the temperature ranging from 100.6 to 104 degrees, and pulse 110 to 120. The spot in the middle lobe of the right lung cleared, but the upper lobe of the right lung again became involved. The course was not progressing satisfactorily.

As the patient was watched, it was difficult to explain why the patient was so seriously sick with so few localized symptoms. Widal and undulant tests were negative.

Upon entering the house to visit the patient on the first day, I saw a pair of love birds and asked if the birds had been in good health (as I have some birds at home and am rather fond of them). I understood the answer to be in the negative.

On further questioning, the following facts were brought out. The birds were bought from a peddler on December 5, 1931. One of these birds did not appear well, was gapping and dumpy. The next day the peddler took that bird back and gave her another bird. One of these birds died shortly, and was replaced through a local dealer. The peddler was peddling by hand, and claimed to have come off a boat at Avila. He was apparently an Australian. The patient's illness began on December 23, 1931, but through false sensitiveness did not tell me she had been under the care of another physician. We killed the two birds and sent them to the laboratory of the University of California.

Laboratory Report.—The report received January 14, 1932, was as follows:

"The examination of the two love birds owned by Mrs. S. has been made. One of the birds showed an enlarged, definitely fatty degenerated liver. Culturally, no organisms were demonstrated except streptococci in the spleen. We have taken some of this material and reinoculated it on other birds and mice. The second bird showed consolidation of the right lobe of the lung. Cultural findings revealed the Friedlander bacillus. Since it is difficult from postmortem examinations to diagnose the existence of psittacosis, we are dependent entirely on the reinoculation of the material which will take some time before we can report on the results thus obtained. In my opinion, bird No. 2 is probably not a case of psittacosis. Bird No. 1 might be considered suspicious, although other infections may cause the same lesions." Signed, K. F. Meyer.

The following is a later report, dated January 27, 1932:

"On January 11 we received from you two shell parakeets or love birds, supposedly owned by Mrs. S. As previously stated, one bird showed an enlarged, fatty, degenerated liver, which was found to be culturally sterile. The reinoculation of this material produced typical psittacosis in rice birds. Passage through mice and the demonstration of typical L-CL-L bodies found in smears from the spleen support the diagnosis and leave no doubt that one bird at least died of psittacosis." Signed, K. F. Meyer.

A report dated February 1, 1932:

"I wish to thank you for your letter of January 27. The specimen of blood was received today and we will subinoculate it into other animals and make an agglutination test as well. Doctor Hazeltine and I concluded that this is probably a mild case of psittacosis." Signed, K. F. Meyer.

And a report from the University of California laboratory, dated February 4, 1932:

B. abortus, negative; ***B. melitensis,*** negative; ***B. typhosis,*** negative; ***B. para*** A, negative; ***B. para*** B, negative. Wassermann, negative.

In our own laboratory the blood culture was negative; sputum was negative for the bacillus of tuberculosis; urine was negative except for one plus acetone and a lowering of the chlorids, at the onset of the illness. On February 8, some pus, blood cells, bacteria, and many uric acid crystals were noted. Blood count on January 29: Red blood cells, 3,480,000; white blood cells, 15,300; hemoglobin, 75 per cent; polymorphonuclears, 72 per cent; small lymphocytes, 18; and large lymphocytes, 10.

On March 4, 1932: red blood cells, 5,000,000; white blood cells, 11,200; hemoglobin, 85 per cent. On January 29, 1932, the blood pressure was 140 systolic, 86 diastolic; weight was 159¼, a loss of over twenty pounds. On March 4, 1932, the blood pressure was 160 systolic, 100 diastolic.

About January 10 the patient was put on quinin and a chlorin mixture, and there was marked improvement. The mental symptoms cleared, temperature dropped to normal, and pulse was 82 to 90. The appetite improved some, but the patient was very weak and had poor color. On January 29, a little fever showed, which ran from 100 to 101.08 degrees on February 3, but disappeared by February 5. There has been no subsequent rise in temperature. The recovery has been extremly slow.

The very marked relapse in this case shows a striking similarity to Case No. 3 in the Peterson, Spaulding, and Wildman's series, printed in the *Journal of the American Medical Association* of July 19, 1930.

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The Function of the Graduate School of the Training of Specialists.—Proper survey of clinical material at the command of the medical school almost always reveals a considerable amount, the study and care of which is not properly a function of the student at the undergraduate level or of the intern preparing for general practice. I suspect that much of the deplorable tendency of so many medical graduates to rush early into the practice of medical specialties results from their enforced contacts as clerks and interns with clinical material which should be reserved for the training of specialists. Much also of the overcrowding of the undergraduate medical curriculum results from the cosmic urge of the specialists in the medical faculty to tell all they know, a desire which might be less harmfully if not more profitably granted if their efforts were directed toward graduate students of clinical specialties.—*Journal of the American Medical Association,* April 30, 1932.